The Diagnostic and Statistical Manual IV, which had been in use from 1994 to 2013 (with a text revision in 2000), classified Posttraumatic Stress Disorder as an Anxiety Disorder. The Diagnostic Statistical Manual V, released on the 18th of May 2013, classifies it as a Trauma and Stressor Related Disorder. This disorder has a history that stretches back to ancient Greece. However, only within the last few decades and due to recent medical advances have we been able to pinpoint specific neurological changes in PTSD patients. The revision in DSM-5 classification is important because it focuses on stress and trauma, two conditions that affect our nervous system, and in some cases, cause significant and irreversible changes. There are three factors that must be considered in the "interplay between adverse environmental stimulation, stress responses/reactions, and pathology" (Sherin and Nemeroff). Those include endocrinology, neurochemistry and brain circuitry of patients with PTSD. To comply with the length requirement of this paper, I will focus only on brain circuitry.

According to J. Douglas Bremner, MD, the regions of the brain that play an important role in PTSD include hippocampus, amygdala, and medial prefrontal cortex. The hippocampus is a part of the limbic system of the brain that is in charge of storing and retrieving memories. "People who have experienced some kind of damage to their hippocampus experience difficulties in or the complete inability to store and recall information" (Tull). Matthew Tull, PhD also states that, along with other limbic structures, hippocampus plays a role in a person's ability to overcome fear responses. A study published in the Medical Science Monitor states that lab rats having received 20 electric foot shocks for a total of 6 times in 3 days showed "significant damage in the hippocampus" (Gao, Wang et al.). According to Bremner, a hallmark feature of PTSD is

reduced hippocampal volume. The prolonged exposure to stress leads to “reduction of dendritic branching, loss of dendritic spines, and impairment of neurogenesis” (Bremner). Amygdala is a structure in the limbic system that is linked to emotion and aggression. It controls fear responses, secretion of hormones, arousal, and the formation of emotional memories. An article published in May this year in BioMed Research International Journal shows that, “the results of a functional neuroimaging meta-analysis in participants with PTSD found evidence of amygdala abnormalities, particularly in the left amygdala, where two distinct clusters of abnormal function were identified: a ventral anterior hyperactivation cluster and a dorsal posterior hypoactivation cluster” (Starcevic, Postic et al.). The same study showed the left amygdala significantly smaller than the right in individuals suffering from PTSD. Furthermore, “In PTSD, limbic biases towards survival-related stimuli are out of balance, and can prime attentional systems to be overly attuned to minor, often irrelevant signals of danger” (Scaglion and Lockwood). The medial prefrontal cortex is in charge of our stress responses and emotional reactivity. It consists of anterior cingulate cortex, subcallosal cortex, and the medial frontal gyrus. According to Radley, Arias and Sawchenko, increasing evidence suggests that medial prefrontal cortex plays an important role in regulating the hypothalamo–pituitary–adrenal (HPA) response to emotional stress. “Functional imaging has found decreased activation of the medial prefrontal cortex in PTSD patients in response to stimuli” (Sherin and Nemeroff). Sherin and Nemeroff also state that patients with PTSD show decreased volumes of the frontal cortex, and well as decreased volumes of anterior cingulate cortex. The studies done on the hippocampus and amygdala of individuals with PTSD cannot

confirm that these structural changes were not in some way present before the trauma occurred. It is impossible to determine whether the hippocampus is directly affected by the trauma or whether its decreased volume "might be a pre-existing vulnerability factor for developing PTSD" (Sherin and Nemeroff). However, in the case of abnormalities in prefrontal cortex, a twin study showed that "unlike the hippocampus, volume loss in anterior cingulate cortex is secondary to the development of PTSD" (Sherin and Nemeroff). To sum up, PTSD patients show exaggerated amygdalar responses, an impaired ability to recognize a threat due to hippocampal and amygdalar dysfunction and a definite lack of the ability to suppress stress responses and fear associations due to abnormal prefrontal function.

In conclusion, the effects of PTSD on Central Nervous System are real, well documented and impossible to ignore. It was discouraging and frustrating to find out that many people still believe PTSD to be a disorder that stems from the 'lack of moral fiber.' The term Posttraumatic Stress Disorder only came in use during mid-1970's, as a result of approximately "30% of Vietnam War veterans" suffering from this disorder (US Department of Veterans Affairs). The more recent discoveries, as well as the change in DSM-5 classification, appear to reflect a concern for not only the psychological symptoms but the neurobiological symptoms as well. With "11-20 Veterans out of 100 who served" in Iraq and Afghanistan having been diagnosed with PTSD, I sincerely hope that this extensive research will not be in vain and that some reversal of these neurobiological changes is possible (US Department of Veterans Affairs).